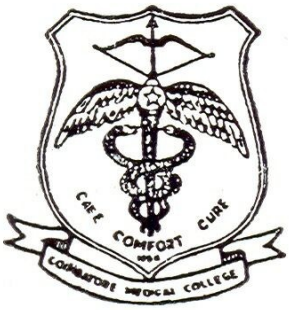
"A STUDY OF VARIOUS PANCREATIC LESIONS"

[An analytical study of 45 cases]



Dissertation submitted to

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M.S. Degree in General Surgery

Branch I

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI.

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COIMBATORE MEDICAL COLLEGE

COIMBATORE – 641 014.

CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. R. Krishna Kumar** and submitted in partial fulfillment of the requirements for the Degree of M.S. General Surgery, Branch -I of the Tamil Nadu Dr. M.G.R. Medical University.

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DECLARATION

I solemnly declare that the dissertation titled **" A STUDY OF VARIOUS PANCREATIC LESIONS"** was done by me at Coimbatore Medical College and Hospital, Coimbatore, during the period of May 2004 to January 2006 under the guidance and supervision of **Prof. Dr. G.S. Ramachandran,** M.S., MNAMS..

The dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of **M.S. DEGREE BRANCH – I IN GENERAL SURGERY.**

PLACE :

DATE : **DR. R.KRISHNAKUMAR**

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It gives me immense pleasure to express my deep sense of gratitude to my chief **Prof. Dr. G.S. Ramachandran, MS, MNAMS** for his able guidance continuous supervision, constant help and encouragement during the course of my study and preparation of this dissertation.

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I wish to express my gratitude to all my Assistant Professors and the rest of the surgical faculty, who helped me to complete this work.

Last but not least, I am grateful to all the patients without whom this work would not have been completed.

Coimbatore

04.02.2006

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Introduction

INTRODUCTION

The pancreas cuddles the left kidney, tickles the spleen. hugs the duodenum, cradles the aorta, opposes the inferior vena cava, dallies with the right renal pedicle, hides behind the posterior parietal peritoneun of the lesser sac and wraps itself around the superior mesentric vessels.

Historically, the first description of the pancreas is credited to Herophilus of Chaikaldon around the year 300 B.C. Four centuries later in approximately 100 A.D this abdominal organ was named the pancreas by Rufus of Ephesus. The first operative intervention on the panrceas has been attributed to Le Dentu in the year 1862, involving percutaneous aspiration of a pancreatic mass with an unfavourable outcome. The first successful resection of a periampullary tumour performed by Halsted in 1899. The tumour was resected locally and reconstruction was performed. The first successful pancreatico duodenectomy was performed by Kausch in 1912. In 1935, **Whipple** did a successful two-stage enbloc resection of the head of the pancreas and the duodenum. The first one stage pancreatico duodenectomy was reported in the United States by Trimble in 1941.

Surgeries in pancreas were considered at one point of time

equivalent to disturbing a sleeping tiger. But the scenario today is entirely different.

The better understanding of anatomy and modern investigation have made surgeries of pancreas a child's play under competent hands.

Even though the incidence pancreatic diseases are not uncommon in the surgical side, the morbidity and mortality in relatively high when compared with any other organic diseases.

As the incidence of alcoholism is alarmingly increasing, the disease of pancreas is also expected to be more prevalent. Hence we decided to study this topic in detail in various aspects such as incidence; surgeries and various modalities of treatment.

Aim of Study

AIM OF STUDY

The aim of this study was to evaluate the etiopathogeneis of various pancreatic pathologies ranging from inflammatory lesions to malignancies and categorizes the various modalities of treatment and their outcome.

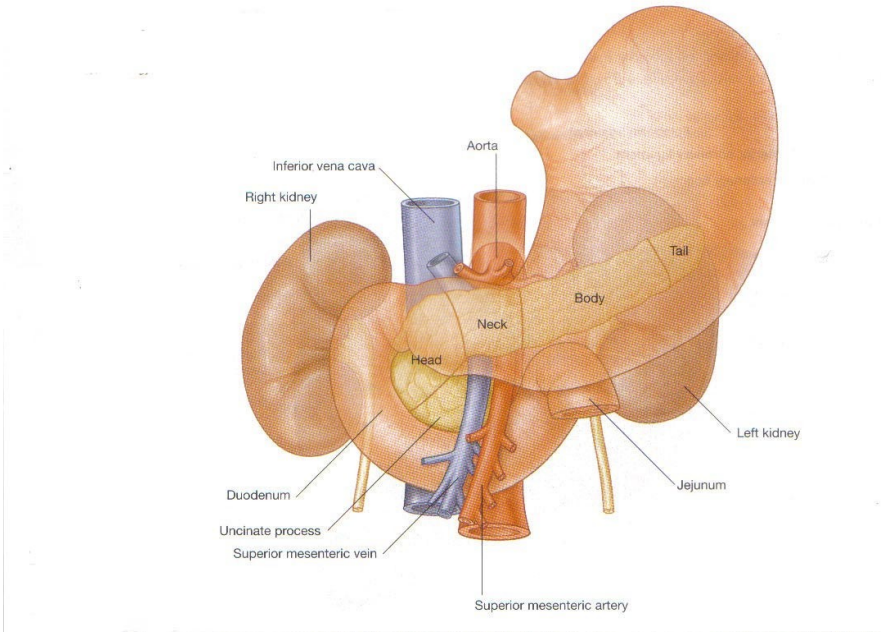
Materials and Methods

MATERIALS AND METHODS

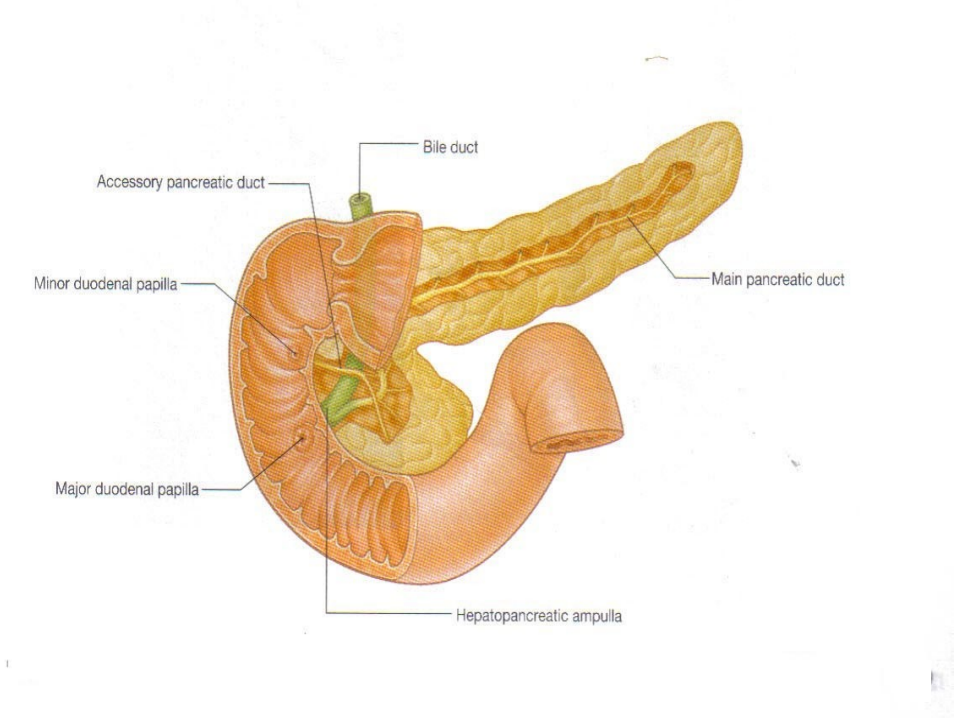
This study consists of all patients admitted in surgical and Gastro enterology ward of Govt. Medical College Hospital, Coimbatore, from

May 2004 to Jan. 2006. Once the patient is admitted the name, age, sex are noted. Clinical evaluation is done in a systematic way. Appropriate investigations are carried out. In those who are operated, the operative findings & methods of management are recorded. Cases are followed up till their discharge from the hospital. Above facts are recorded in a proforma prepared for this study.

Surgical Anatomy of Pancreas



ANATOMY AND RELATIONS OF PANCREAS



MAJOR AND MINOR DUCTS OF PANCREAS

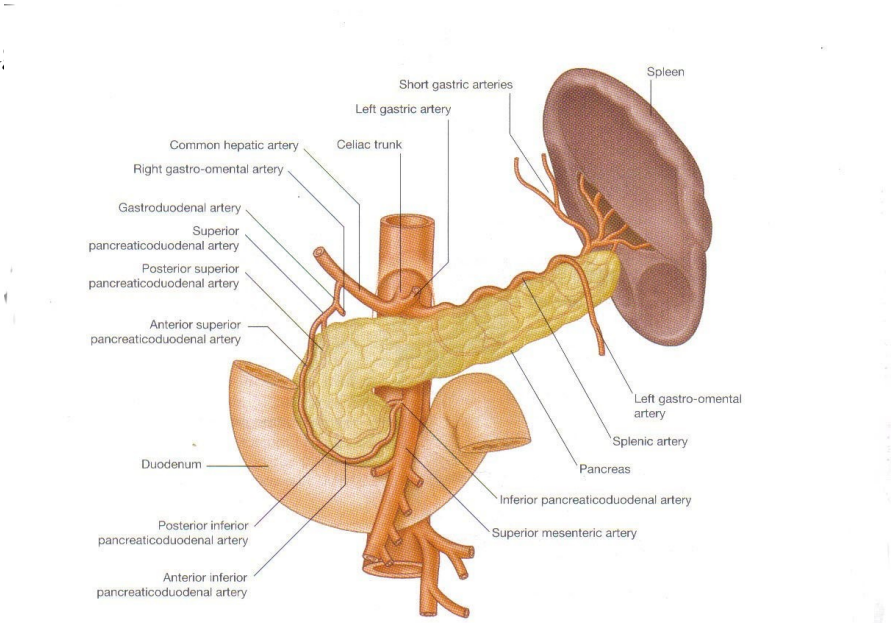
SURGICAL ANATOMY OF PANCREAS

A revision of the surgical anatomy[1,2,3,4,5] of the pancreas is necessary at this juncture to appreciate the various aspects of surgeries of pancreas. Pancreas lies transversely across the upper part of the posterior

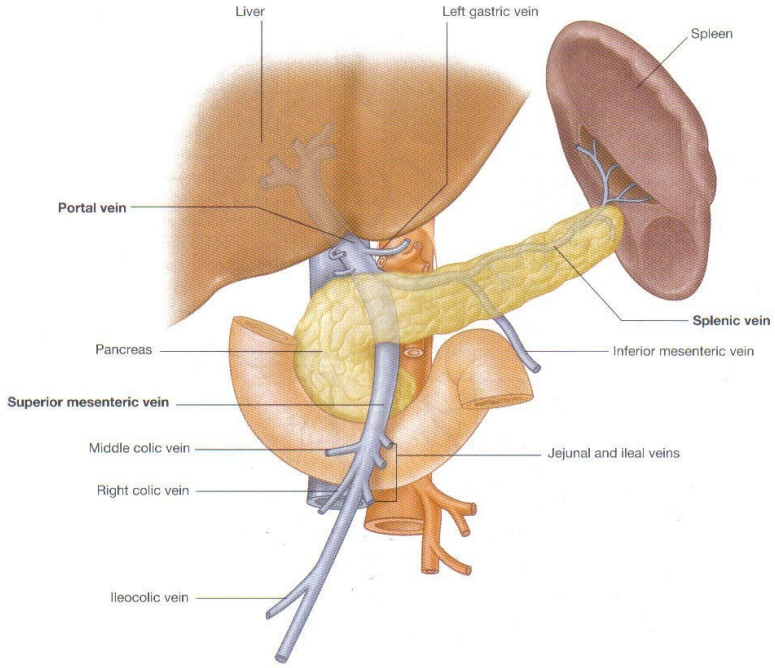
abdominal wall and is about 15-20 cm in length. 3.1 cm in width; 1-1.5 cm in thickness and weighs about 80-90 gms.

Posterior to the pancreas is the inferior vena cava, Aorta, Lt kidney, both renal veins and right renal artery. Pancreatic head lies with in the concave sweep of the duodenum. Body crosses the spine and is directed somewhat obliquely and superiorly to the left with the tail residing in the hilum of the spleen. Splenic artery runs along the upper border of the pancreas, and the Splenic vein runs behind, just superior to the lower edge. The superior mesentric vein and artery lies just behind the neck of pancreas and are also enclosed posteriorly by an extension of the head known as uncinate process. The uncinate process lies between the inferior vena cava and the portal vein.

The main pancreatic duct of Wirsung usually traverses the entire length of the gland slightly above a line halfway between the superior and inferior ed



ARTERIAL SUPPLY OF PANCREAS



VENOUS DRAINAGE OF PANCREAS

accessory duct of Santorini branches out from the pancreatic duct in the "neck of the pancreas and empties into the duodenum, about 2.5 cm above the duodenal papillae.

THE ARTERIAL AND VENOUS BLOOD SUPPLY

Head and neck of pancreas are supplied by branches from the anterior and posterior pancreatico - duodenal arcades, which are formed by the union of superior and inferior pancreatico - duodenal arteries. Superior pancreaticoduodenal artery is a branch of gastro-duodenal trunk which itself is a branch of right hepatic artery. The inferior pancreatico - duodenal artery is a branch from the superior mesentric artery.

The body and tail of pancreas are supplied by a number of branches arising from the spleenic artery viz-dorsal pancreatic artery, inferior pancreatic artery, arteria pancreatica magna and caudal pancreatic arteries.

VENOUS DRAINAGE

Is into the portal, spleenic and superior mesentric vein.

LYMPHATIC DRAINAGE

Lymph capillaries commence around the acini and their continuations following the blood vessels: There are no lymphatics in the pancreatic islets. Most vessels end in the pancreatico - spleenic nodes. Some in nodes along the pancreatico - duodenal vessels, and

others in the superior mesentric pre - aortic nodes.

Surgical Physiology of Pancreas

SURGICAL PHYSIOLOGY OF PANCREAS

In response to a meal, pancreas secretes[6,7] digestive enzymes in an alkaline (P.H.8.4) bicarbonate rich fluid. Acinar cells synthesize and secrete digestive enzymes, while the duct cells secrete bicarbonate. Daily

secretion is about one liter containing 5-8 gm of protein in the form of enzymes. About 20 digestive enzymes are produced.

Proteolytic enzymes – eg; trypsin

Lipolytic enzymes – eg; lipase

Starch splitting enzymes – eg; Amylase

Nucleic acid splitting enzymes – eg; ribonuclease

Pancreatic enzyme secretion is potently stimulated by Pancreozymin, which is released from the duodenal mucosa by luminal fat and peptides.

Rate of secretions and the bicarbonate content of the pancreatic juice is increased by hormone SECRETIN that is released from the duodenal mucosa by luminal acid stimulation.

Pancreatic secretion can also be stimulated by hormones produced with in the pancreas and small bowel, vasoactive intestinal peptide, (VIP) and gastrin and inhibited by the pancreatic hormones, somatostatin, pancreatic polypeptide and glucagon.

Pancreatic secretions consist of a CEPHALIC PHASE - initiated by thought of food, a GASTRIC PHASE produced by food in the stomach and an INTESTINAL PHASE mediated by secretion of Pancreozymin released from the duodenum and Jejunum.

Histology of Pancreas

HISTOLOGY OF PANCREAS

Pancreas[8] has exocrine and endocrine parts. Exocrine pancreas is in the form of a serous, compound tubulo-alveolar gland. Gland surrounded by a delicate capsule. Septa extend from the capsule into the gland and divide it into lobules. The secretory elements of the exocrine pancreas (They are usually described as alveoli) are long and tubular. The lining

cells appear triangular in section and have spherical nuclear located basically. In addition to secretary cells, the alveoli of the exocrine pancreas contain centroacinar cells that are so called because; they appear to be located near the centre of the acinus (or alveolus).

Endocrine part of the pancreas is in the form of numerous rounded collections of cells that are embedded with in the exocrine part; the human pancreas has about 1 million islets. Each islet is separated from the surrounding alveoli by a thin layer of reticular tissues. Three main types of islets cells are α cell, β cell and δ cells, which secretes glucagons, insulin and somatostatin respectively. In islets of human pancreas, the α cells tend to be arranged towards the periphery (or cortex). In contras, the β cells tend to be lie near the centre (or medulla) of the islets. δ Cells are also peripherally placed.

GROSS APPEARANCE OF ACUTE PANCREATITIS

PATHOLOGY OF PANCREATIC DISEASES

The most significant disorders of the exocrine pancreas[9,10,11] are cystic fibrosis, acute and chronic pancreatitis and tumours.

In cystic fibrosis, pancreatic abnormalities present in

approximately 85 to 90% of patients. In milder cases there may be only accumulations of mucous in the small ducts with some dilatation of the exocrine glands. In more advanced cases, usually seen in older children or adolescents, the ducts are totally plugged, causing atrophy of the exocrine glands and progressive fibrosis.

In acute pancreatitis four basic alterations are seen histologically 1) Proteolytic destruction of pancreatic substance, 2) necrosis of blood vessels with subsequent hemorrhage, 3) necrosis of fat by Lipolytic enzymes and 4) an accompanying inflammatory reaction. These alterations are depending on the duration and severity of the diseases. Acute pancreat on of fat cells in which shadowy outlines of cell ipitate.

GROSS APPEARANCE OF CHRONIC PANCREATITIS

Macroscopically characteristics of acute pancreatic necrosis are areas of grey white Proteolytic destruction of parenchymal substance, hemorrhage and chalky white areas of fat necrosis. Characteristically, the peritoneal cavity contains a serous and slightly turbid, brown tinged fluid in which globules of oil can be identified. (So called chicken broth fluid) Foci of fat necrosis may be found in fat depots of omentum, mesentry of

bowel and properitoneal deposits. If the patients survives the acute necrotizing damage may resolve slowly and be replaced by diffuse (or) focal parenchymal (or) stromal fibrosis, calcifications and irregular ductal dilatations occasionally liquefied areas are walled off by fibrous tissue to from small or large cystic spaces known as PSEUDOCYSTS.

The most common type of chronic pancreatitis is chronic calcifying pancreatitis mostly seen in alcoholics. All components of involved lobules are affected. There is atrophy of the acini, marked increase in interlobular fibrous tissue and a chronic inflammatory infiltrate around lobules and ducts. The interlobular and intralobular ducts are dilated and contain protein plugs in their lumina. The ductal epithelium may be atrophied (or) hyper plastic (or) may show squamous metaplasia. Grossly the gland is hard and exhibits foci of calcification and fully developed pancreatic calculi. Pseudocyst formation is common in this type of

GROSS APPEARANCE OF INVASIVE DUCTAL CARCINOMA OF PANCREAS

Another type is chronic obstructive pancreatitis, the distribution of lesion is not lobular and the ductal epithelium generally is less severely damaged. The most common cause of this type is stenosis of the sphincter of oddi, associated with. Cholelithiasis. The lesions are more prominent in the head of pancreas.

All Ca are arising from ductal epithelium. Carcinomas head of the pancreas are fairly small lesions. Some lesions up to 8 to 10cm in

diameter.

The grey, white, scirrhous homogenous Tumour infiltrates and replaces the lobular architecture of a normal pancreas. Such lesions have poorly defined, infiltrative margins, extents in to the margin of the duodenum & CBD, Sometimes it produce either a small fungating lesion or an irregular ulceration. In this infiltrative growth, it surrounds and compresses the CBD (or) ampulla of vater causing biliary obstruction. Extensions to porta hepatic nodes are also common.

Ca of body & tail of pancreas are unusually large, hard and irregular mass. On cross section, it resembles the Ca head of pancreas but frequently extend more widely than those of the head. They invade the adjacent vertebral column, retroperitoneal space; spleen (or) adrenal, transverse colon & stomach Massive hepatic metabolism are quite common of Ca of tail and body of pancreas.

Histologically more or less well differentiated glandular patterns and is thus adenocarcinomas. The tumours may be mucinous or non-musin secreting. The glands are atypical, irregular, small and are usually lined by anaplastic cuboidal to columnar epithelial cells. About 10% either adenosquamous pattern (or) giant cell formation. 0.5% -Cystadenocarcinoma arises in cysts. Rarely acinar cell ca - arise form

acinar cells.

Pancreatic cysts have been classified as True cysts and false cysts (Pseudocyst) True cysts contain epithelial lining where as false cysts do not have epithelial lining. Congenital cysts (or) true cysts usually multiple but occasionally occur single. They range in size from microscopic lesions to large spaces up to 3 to 5 cm in diameter. They are lined by smooth, glistening membrane lined by atrophied epithelial cells (or) low cuboidal epithelial cells. They are usually enclosed in their fibrous capsule and are filled with a clear to turbid mucoid (or) serous fluid

Pseudocyst is collection of fluid that arises from loculation of inflammatory processes, necrosis (or) hemorrhages. These are clinically important always associated with pancreatitis secondary to alcohol abuse, biliary tree disease (or) trauma. These are usually solitary and measures 5 to 10 cm in diameter. Cysts wall may be thin (or) thick and fibrous. They do not have an epithelial lining and have no connection (or) communication with surrounding ductal system. There may be a marked inflammatory reaction in the fibrous capsules. Cystic fluid is usually serous and turbid. But the Pseudocyst occurs as a result of chronic pancreatitis is of retention type and the duct is decreased and strictured. Duct - cyst communication always present.

Cystic pancreatic neoplasm consist about 10% - 20% of all cystic lesions. Depending on their serous (or) mucinous component they have been classified into benign and malignant. Mucinouscystadenoma tend to progress to malignancy and serous cystadenoma can be considered benign. Papillary cystic tumour, cystic islet cell tumour and acinar Cystadenocarcinoma are other cystic neoplasm. Some may undergo necrosis and cystic degeneration, can present as a cystic mass and these include ductal adenocarcinomas, sarcoma & lymphomas.

Calcification of the rim of cyst wall is common in cystic tumour. Central 'Sun Burst' calcification is reported to be highly suggestive of serous cystadenoma 'and also occurs in 33% of mucinous adenocarcinomas.

MY STUDY

INTRODUCTION

I have studied 45 cases of pancreatic diseases. The break up 45 pancreatic diseases is as follows.

Acute pancreatitis	-	7 cases
Pseudocyst of pancreas	-	15 cases

Chronic calculus pancreatitis - 12 cases

Ca. head of pancreas - 11 cases

The age and sex incidence of pancreatic disease as follows

Age	**Sex**	
	M	**F**
10 – 20	2	2
21 – 30	7	0
31 – 40	13	2
41 – 50	5	3
51 – 60	3	3
More than 60	2	3
	32	+ 13 = 45 cases

Clinical Presentation

CULLEN SIGN

CLINICAL PRESENTATION

ACUTC PANCREATITIS

Patent with acute pancreatitis are mostly presented with pain abdomen (>70%). Pain was acute in onset and presented with shock. Pain mostly in mild epigastric region and also some patients presented with fever nausea, vomiting, and hiccup. Most of the patients were chronic alcoholic and less than 40 years old. On examination patient was in

shock. There was tenderness in the mild epigastric region. Abdominal distension was also present.

CHRONIC PANCREATITIS

Patient with chronic pancreatitis were mostly presented with pain abdomen and also pain was referred to back and left & Right hypochondrium. Most are suffered from malabsorption and weight loss. Most patients were chronic alcoholic. On examination, tenderness over epigastric region was present.

PSEUDOCYST OF PANCREAS

Patients with Pseudocyst of pancreas were mostly presented with pain abdomen, anorexia and vomiting. One patient presented with epigastric mass. 99% patients were chronic alcoholic, one patient developed Pseudocyst of pancreas following a blunt injury abdomen and

PATIENT WITH PSEUDOCYST

seven patients developed Pseudocyst of pancreas following an attack of acute pancreatits. Patients with cysts in the pancreas due to chronic calculus pancreatitis were also presented with abdominal pain, nausea, and vomiting.

CA HEAD OF PANCREAS

Patients with carcinoma head of pancreas were mostly presented with Jaundice, weight loss, anorexia and pain abdomen. One patient presented with abdominal distension and engorged vein over anterior abdominal wall. One patient presented with progressive jaundice with severe anorexia. Two patients were chronic alcoholic and chronic smoker.

One patient present with diabetes mellitus and another patient present ·with pedal oedema and dyspnoea. On examination some patients have epigastric mass and some have liver and palpable gall bladder.

Total No of Cases: 11

Symptoms	No. Of Cases	Percentage
Jaundice	8	73%
Weight Loss	9	82%
Pain Abdomen	6	55%
Anorexia	9	82%
Ascites	2	20%

Investigations

INVESTIGATIONS

All the patients presented with pain abdomen and with a history of chronic alcoholism are evaluated by the estimation of serum amylase level. It was found minimum elevation to very high level ranging from 180 U/L to 1544. U/L. All the patients were evaluated for altered liver function tests. Routine blood investigations were also done.

RADIOLOGICAL INVESTIGATIONS

For all the patients with pain abdomen and chronic alcoholism and jaundice. Plain X-ray abdomen was taken. Six of the patients showed areas of calcification along the line of pancreas of varying size.

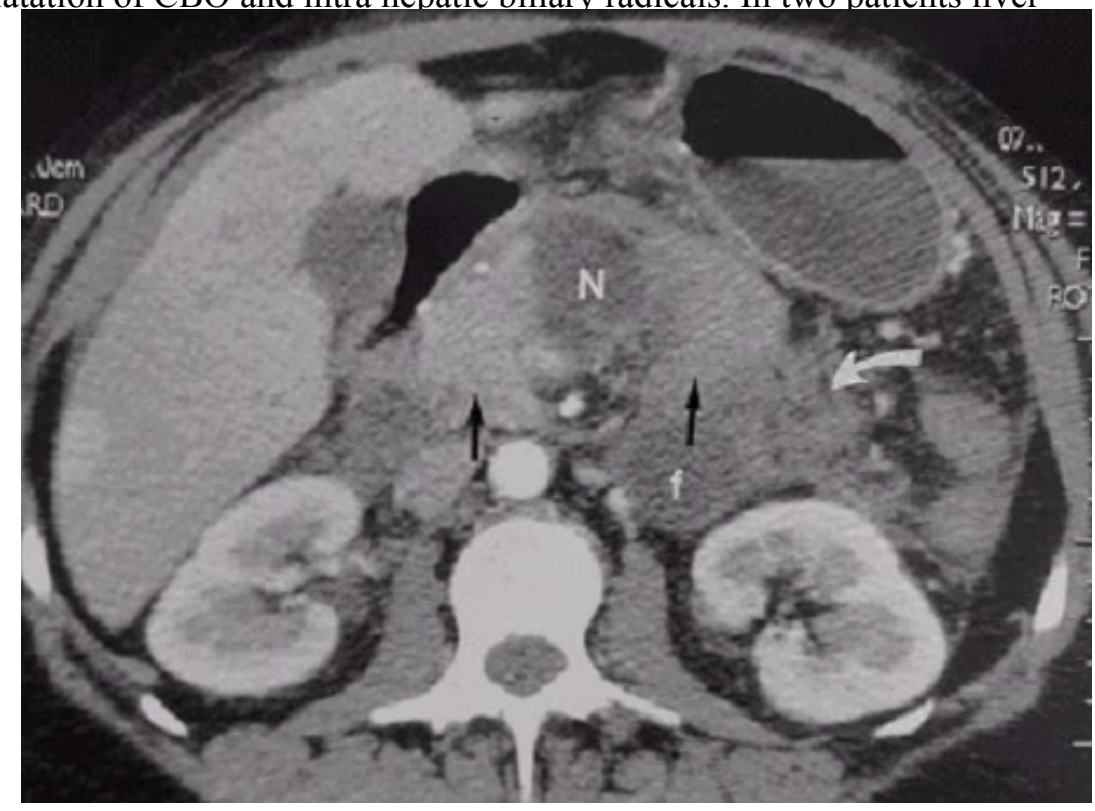
ULTRA SONOGRAPHIC EXAMINATION

Pseudocyst of pancreas

Most of the cysts were situated in the head of pancreas. Sizes of the cysts were ranging from 4cm x 4cm to a maximum of 20cm x 15cm. This large cyst was found in a patient with a history of blunt injury abdomen for that laparotomy has been done.

Ca Pancreas

In 11 patients of Ca pancreas some have mass in head of pancreas with liver enlargement and GB with thickened wall. They also showed dilatation of CBO and intra hepatic biliary radicals. In two patients liver



CT SCAN FINDING OF PERIPANCREATIC FAT NECROSIS

secondaries with lymph nodal metastasis along greater curvature and pancreatic duodenal nodes were also present. In one of the above two patients hemorrhagic ascites features were also seen.

Chronic Calculus Pancreatitis

In chronic calculus pancreatitis patients, the ultra sonographic findings were pancreas with multiple large calculi packed in the entire

length of the pancreatic duct of size varying from 1.5cm to 2cm diameter. Pancreatic duct was dilated. Gall bladder was distended with dilated intra hepatic biliary radicals.

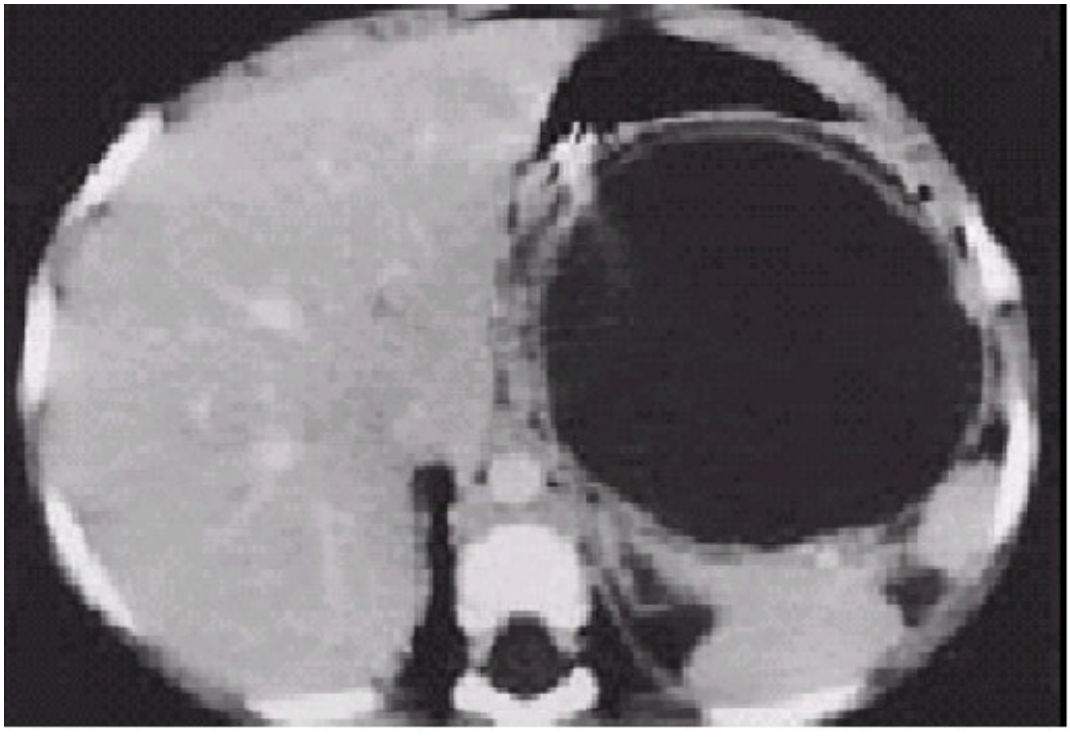
Acute Pancreatitis

In acute pancreatitis patients the ultra sonographic findings was edematous pancreas. Areas of parenchymal necrosis were present.

CT SCAN

Ca Head of Pancreas

CT Scan finding of the Ca Head of pancreas was growth in the head of pancreas with intra hepatic and extra hepatic biliary radicals dilatation. No Para aortic & porta hepatic nodal involvement. In .two patients multiple liver secondaries and Para aortic nodes were present.



CT SCAN FINDING OF PSEUDOCYST

Chronic Calculus Pancreatitis

CT scan findings of calculus pancreatitis were calculi in the pancreatic duct with dilatation of pancreatic duct. In one patient cystic degeneration of parenchyma was present.

Pseudocyst of Pancreas

CT scan findings of Pseudocyst of pancreas were enlarged

pancreas and cystic lesion of varying size mainly in the head of pancreas and some in the body and tail of pancreas.

Acute Pancreatitis

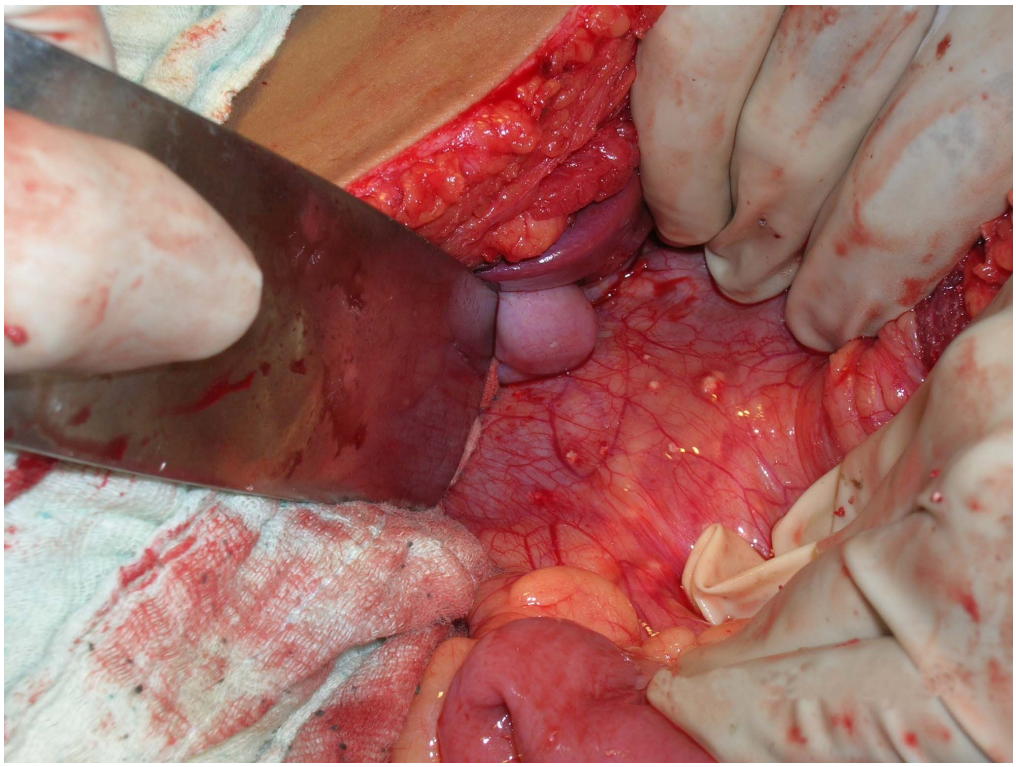
CT scan finding of acute pancreatitis was mostly hemorrhagic edematous parenchyma and some areas of necrosis present.

Percutaneous Transhepatic Cholangiogram (PTC)

PTC finding of one patient was dilatation of intra hepatic biliary radicals and hepatic ducts with obstruction of common hepatic duct - - - - - - ?Malignant growth.

OGD finding of one patient had extra luminal compression.

Management



ACUTE PANCREATITIS AT LAPAROTOMY

MANAGEMENT

Acute Pancreatitis

Out of Seven cases of acute pancreatitis three patients were treated conservatively by the following regimen.

1. IV Fluids
2. Ryle's tube aspiration
3. Analgesics
4. Antibiotics

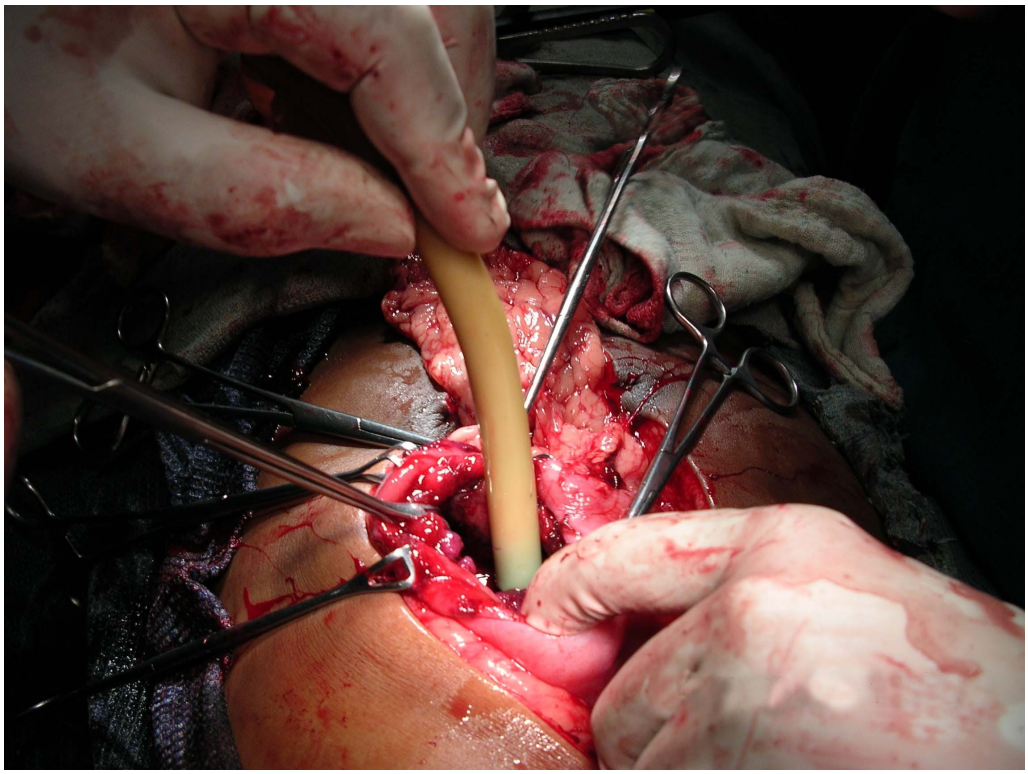
5. H_2 receptor antagonists
6. Sedatives

Another four patients were treated surgically in the form of laparotomy followed by peritoneal lavage with placement of bilateral flank drainage tubes.

Pseudocyst of Pancreas

Out of 15 cases of pseudopancreatic cysts, 12 cases has been treated conservatively by the following regimen, because of small size of cysts

1. IV Fluids
2. Ryle's tube aspiration
3. Analgesics



CYSTO GASTROSTOMY

4. Antibiotics
5. H_2 receptor antagonists
6. Anti cholinergic (Probanthine)
7. Sedatives

Three cases have been treated surgically. The different surgical procedures adapted were as follows

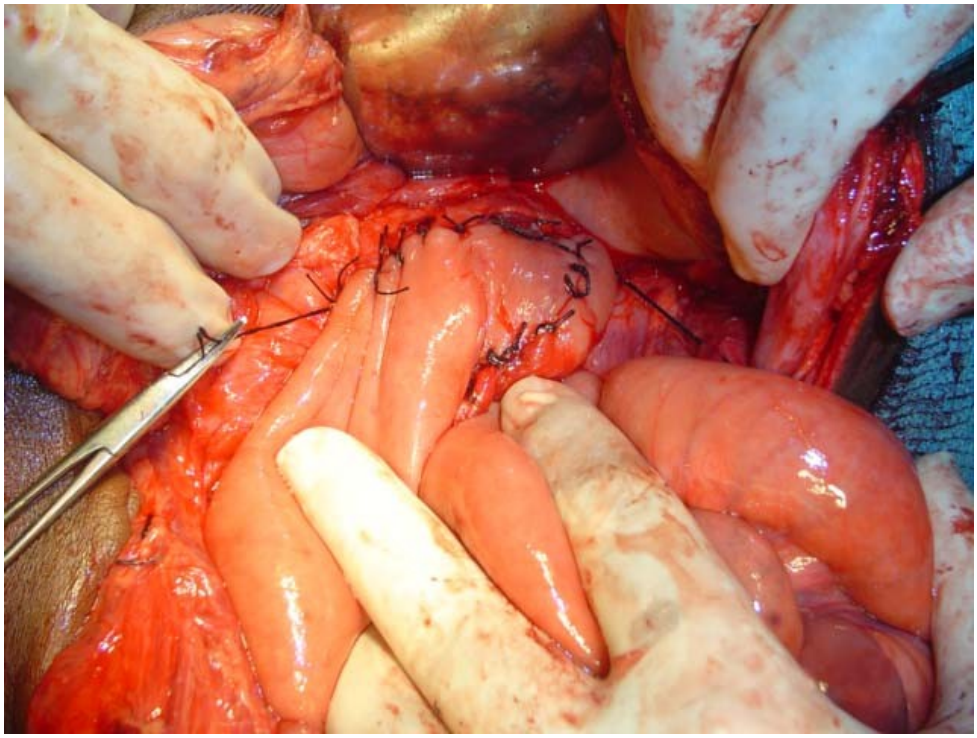
1.Cystogastrostomy for a cyst of size 20 x 15cm situated in the region of head of pancreas.

2.Cystojejunostomy for a cyst of size 15x15cm situated in the region of tail of pancreas occurring as a result of blunt injury abdomen. In this case, 3 to 3.5 liters of necrotic material were aspirated.

3.External drainage of the cyst by a mallecot's catheter as it had ruptured into peritoneal cavity.

Chronic Calculus Pancreatitis

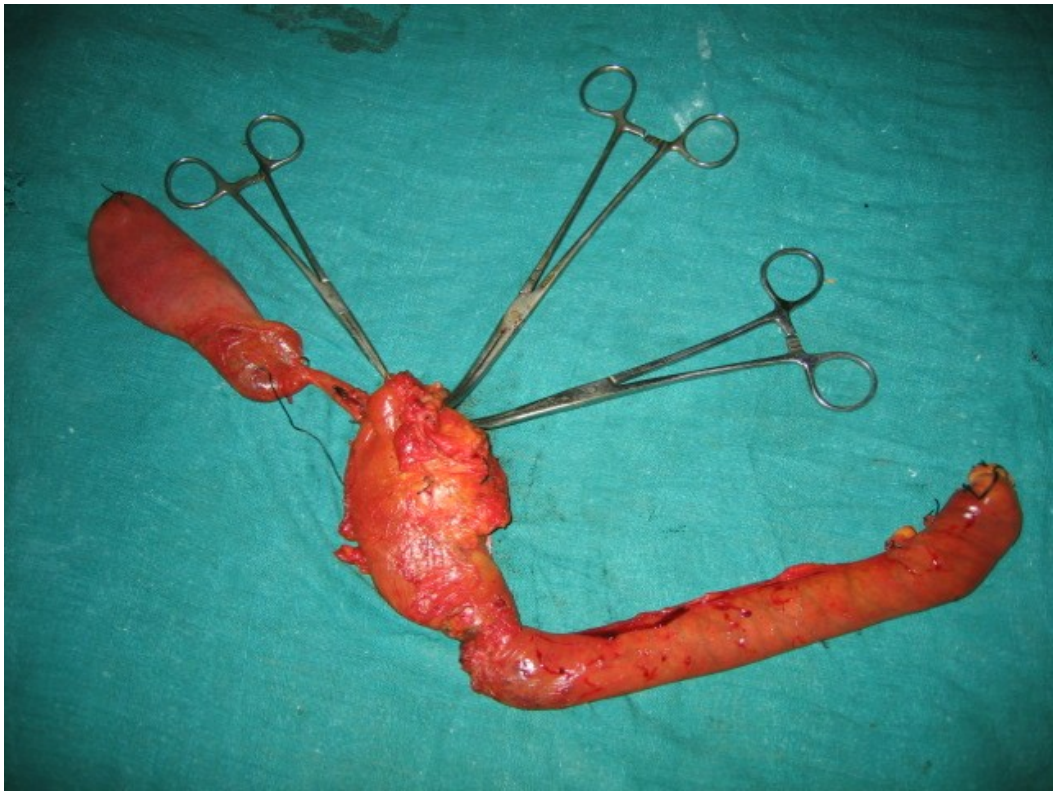
Out of twelve patients of chronic calculus pancreatits, one patient presented with Pseudocyst with calculi in the pancreatic duct. This patient has been treated surgically by pancreatic lithotomy followed by Cystojejunostomy and jejunojejunostomy. Five case of chronic calculus pancreatitis without Pseudocyst were treated surgically as follows:



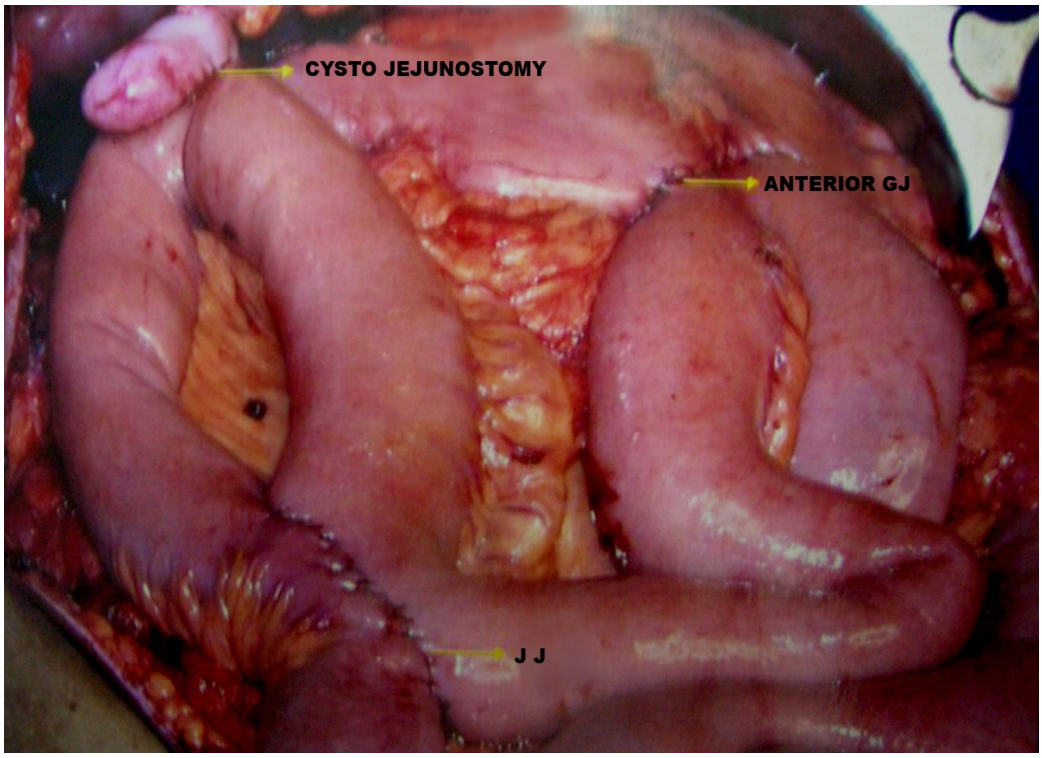
PUESTO PROCEDURE

PANCREATIC CALCULI

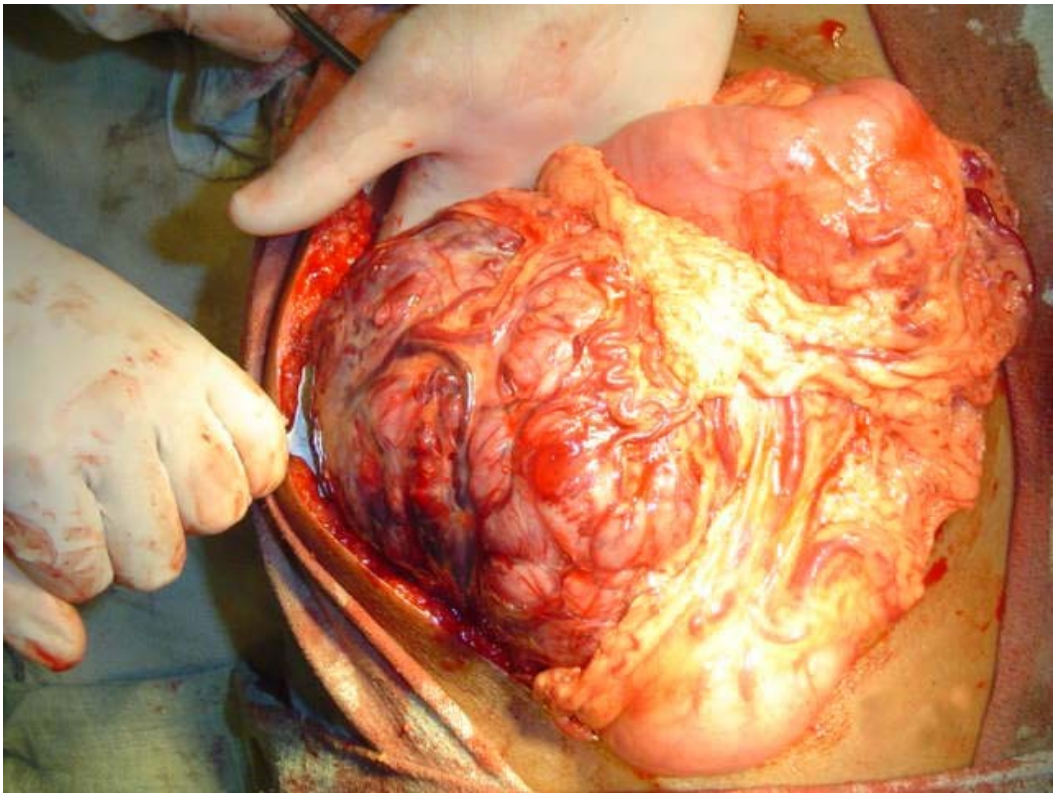
WHIPPLE'S PROCEDURE



RESECTED SPECIMEN WHIPPLE'S PROCEDURE



TRIPLE ANATAMOSIS



HUGE CYSTADENO CARCINOMA FROM HEAD OF PANCREAS

1.Pancreatico-lithotomy followed by pancreatico jejunostomy and jejunojejunostomy - Two' cases.

2.Pancreatico lithotomy followed by Roux-en-Y pancreatico jejunostomy and jejunojejunostomy - One case.

3.Pancreatico lithotomy, pancreatico jejunostomy, choledhoco-duodenostomy, gastrojejunostomy - One case

4.Pancreatico lithotomy, pancreatico jejunostomy, cholecysto jejunostomy, jejunojejunostomy - One case

Ca Head of Pancreas

Out of 11 cases of Ca head of pancreas, six patients were treated conservatively. Four cases underwent radical surgical treatment. They were treated with WHIPPLE'S PROCEDURE Pancreaticodeuodenectomy followed by choledochojejunostomy (cholecysto jejunostomy), pancreatico jejunostomy with gastrojejunostomy.

For three cases, palliative surgical bye-pass procedure done.

For three cases, because of extensive infiltration only biopsy was taken. Post operatively they were treated by chemotherapy.

For one patient chemotherapy was given as per the ultra sonographic and CT scan finding of extensive liver secondaries and ascites.

Discussion and Review of Literature

DISCUSSION AND REVIEW OF LITERATURE

ACUTE PANCREATITIS:

In my study, I have come across 7 cases of acute pancreatits. All the patients were alcoholic. I noticed that Alcohol is the prime etiological factor in acute pancreatitis. The other etiological factors are as follows[12] :

1. Alcoholism
2. Biliary tract disease
3. Trauma
    - Surgical
    - Blunt injury
    - Penetrating injury
    - ERCP
    - Aortography
4. Drugs
    - Thiazides
    - Steriods
    - Azathioprine
    - Frusemide
    - Sulfonamides
    - Clonidine
    - Phenformin
    - Tetracycline
5. Metabolic
    - Hyperparathyroidism

Hyperlipidaemia

Hypercalcaemia

6. Infections

Mumps

Coxsackie - B virus

Mycoplasma pneumonia

Infectious mononucleosis

Septicaemia

7. Congenital mechanical obstruction of pancreatic duct

Pancreas divisum

8. Periampullary carcinoma
9. Hereditary pancreatitis
10. Vascular disease

Cardio pulmonary by pass

Periarteritis nodosa

Athero embolism

It is generally believed that acute pancreatitis is triggered by obstruction of the pancreatic duct and that the injury begins with in the pancreatic acinar cells.

One of the most widely accepted theories is co-localization theory.

Co-Localization Hypothesis[17]

This explains that co-localization of digestive enzymes Zymogens such as trypsinogen with lysosomal hydrolases such as cathepsin- B inside the cytoplasmic vacuoles. Under these conditions cathepsin- B activate the other enzymes. So according to Co-localization hypothesis. Cathepsin- B mediated intra – acinar cell activation of the digestive enzymes leads to acinar cell injury and triggers an intra – pancreatic inflammatory response.

In my study all the patients presented with pain abdomen (100%); vomiting and shock (50%). The other common symptoms were retching and hiccough rarely diarrhea, dyspnoea, cyanosis, haematemesis and melena may appear. In my study, except for epigastric tenderness and shock, no other signs were presented. Other signs were mild jaundice and abdominal distension in early stage. Other late signs were bluish discoloration of the skin around the periumbilical area (CULLEN'S SIGN) or in the loin (GREY TURNER'S SIGN). Rarely polyarthritis (or) bone pain may be observed.

In my study, I came to know that serum amylase estimation is the

diagnostic test for acute pancreatitis. The other tests, which could be done, are

1.Urinary amylase and lipase

2.Serum ribonuclease (or) deoxy ribonuclease

3.Hyperglycemia and glycosuria.

4.Serum Amyloid- A[18]

5.Hypocalcaemia

6.Methaemalbuminaemia

7.Blood coagulation tests (eg) Serum fibrinogen level – elevated

8.C-Reactive protein

9.Hyperlipidaemia

10.Plasma trypsinogen activation peptide (Plasma TAP)[19]

Among the above-sited tests serum lipase estimation is more specific for pancreatic disease than amylase. Because lipase is solely of pancreatic orgin & Serum amylase level may be increased in intra abdominal pathologies like,

Intestinal obstruction

Perforated peptic ulcer.

Acute appendicitis

Ruptured ectopic pregnancy

And extra abdominal pathological conditions like salivary gland

disorders like.

Mumps

Parotitis

Renal failure

Pneumonia

Cerebral trauma

Severe burns

Diabetic ketoacidosis

Amylase – creatinine clearance ratio is also a useful diagnostic tool in acute pancreatitis.

$$\frac{\text{Urine amylase}}{\text{Serum amylase}} \text{ x } \frac{\text{Serum creatinine}}{\text{Urine creatinine}} \text{ x } 100 = \text{the amylase} - \text{Creatinine Clearance ratio}$$

Normally this ratio is 1 to 4%. If it is greater than 6% it indicates acute pancreatitis.

In acute pancreatitis there may be ECG changes also. They are:

1. ST segment elevation (or) depression

2. Inversion of T waves
3. Extended T wave negatively

In acute pancreatits patients, plain X-ray abdomen and chest X-ray may show evidence of pneumoperitoneum. The radiological sings include intestinal distension in the region of the pancreas like

1. Sentinel jejunal loop
2. Colon cut - off
3. Duodenal ileus
4. Generalized paralytic ileus
5. Obliteration of psoas outline
6. Elevation of left diaphragm

Ultrasonography abdomen may be used in the diagnosis of acute pancreatitis. Unfortunately the value of USG is often limited by the presence of air and fluid filled loops of bowel overlying and obscuring the pancreas.

Endoscope Ultrasonography is useful in the evaluation of idiopathic acute pancreatitis[20]. Endoscope Ultrasonography is able to identify significant pathology in pts in whom a diagnosis of 'Idiopathic'

pancreatitis has been made following standard investigation. It can pickup tiny stones in biliary tract, pancreatic duct and gall bladder, which may be missed in routine transabdominal Ultrasonography.

Currently the most widely accepted and sensitive method used to confirm the diagnosis of acute pancreatitis is CT[21]. CT findings in acute pancreatits are

(A) Pancreatic Changes

1. Parenchymal enlargement
   (a) Diffuse
   (b) Focal
2. Parenchymal oedema
3. Necrosis
4. Parenchymal vascular enhancement

(B) Peripancreatic changes

1. Blurring of fat planes
2. Thickening of fascial planes.
3. Presence of fluid collections.

(c) Non specific findings

1. Bowel distension
2. Pleural effusion

3. Mesentric edema

The clinical course in up to 90% of patients with acute pancreatitis follows with mild self-limited pattern. However in 10% to 15% of patients a severe form of illness may occur. It is possible to predict the severity of an attack of pancreatitis objectively by 11 early parameters identified by RANSON in 1974. Most useful in patients with pancreatitis not related to gall stones.

Ransons Criteria[22]

On admission to hospital

1. Age greater than 55 years
2. WBC > 16,000 cells / cumm
3. Fasting Blood glucose > 200mgm / 100ml
4. Serum LDH > 350 IU/L
5. SGOT > 250 U / 100ml

With in initial 48 hrs of admission

6. Haematocrit fall > 10% points
7. BUN elevation > 5mgm / 100ml
8. Serum calcium fall to < 8 mgm / 100ml
9. Arterial Po2 <60mm of Hg
10. Base deficit > 4m Eq / L
11. Estimated fluid sequestration > 6 L

In patients with less than 3 of these 11 signs, the mortality rate is 0.9%.

Less than 3 signs. 0.9%
With 3 – 4 signs. 18%
With 5 – 6 sings 50%
With > 6 signs. 90%

A study[23] from England showed markers of oxidative stress like Ascorbic acid, Thiobarbituric acid reactive substances, Myeloperoxidase detection were highly correlated with severity of pancreatitis.

In my study, out of 7 cases of acute pancreatitis three patients were treated conservatively by the following regimen,

1. IV Fluids
2. Ryle's tube aspiration
3. Analgesics
4. Antibiotics
5. H_2 receptors antagonists
6. Anti cholinergic (Probanthine)
7. Sedatives

Proposed Non operative therapies for acute pancreatits are as follows

Supportive measures

- IV fluid therapy

- Electrolyte replacement
- Analgesics
- Nutritional support
- Antibiotics
- Respiratory support

Pancreatic exocrine secretion suppression

- Nasogastric suction
- Histamine H_2 receptor antagonists
- Antacids
- Anti cholinergics
- Glucagon
- Calcitonin
- Somatostatin

Pancreatic enzyme inhibition

- Protease inhibitors
- Aprotinin
- Fresh frozen plasma
- Antifibrinolytics
- Chloroquine
- Phospholipase A inhibitors

Pancreatic protection from oxygen derived free radicals

- Free radical scavengers
- Xanthine oxidase inhibitors

- Isovolemic haemodilution

Elimination of toxic intraperitoneal compound

- Peritoneal dialysis

Four patients of acute pancreatitis are treated surgically in the form of laparotomy followed by peritoneal lavage with placement of bilateral flank drainage tube.

There are four situations where operative intervention is indicated in a acute pancreatitis patients

1.When the diagnosis is in doubt

2.Patients with known biliary stone disease

3.Failure of patient to improve on medical management

4.Treatment of secondary pancreatic infections like, Pseudocyst formation, abscess formation, Hemorrhage resulting from pseudoaneurysm (or) sectorial (left sided) portal Hypertension.

When laparotomy is performed early in the course of acute pancreatitis, one or more of the following procedures may be advisable[12].

1.Laparotomy alone

2.Placement of catheters for peritoneal lavage.

3.Biliary decompression via a cholecystostomy (or) a T-Tube in the CBD

4.Operative Cholangiogram

5.Cholecystostomy, common bile duct exploration and choledocholithotomy with (or) without an sphincteroplasty.

6.Total (or) sub total pancreatic resection

7.Pancreatic and retroperitoneal debridement and drainage

8.Decompression gastrostomy and feeding jejunostomy.

Two surgical pitfalls in acute pancreatitis are

1. To operate too early and do too much
2. To operate too late and do too little

Early death in acute pancreatitis is rare now days, mainly as a result of modern intensive care treatment. 9 out 10 death[28] occurred more than 3 weeks after the disease onset. Infection of pancreatic necrosis won the main risk factor for death.

Some studies dealt with early central feeding in acute pancreatitis. One study[24] showed supplementary lactobacillus.

Plantarum 299 with fibre diet was effective in reducing the pancreatic sepsis and the number of surgical interventions.

Another study[25] showed early entral nutrition did not ameliorate the inflammatory response in patients with prognostically severe acute pancreatitis. It did not have a beneficial effect on intestinal permeability.

An article[26] from American journal of surgery Glucagon like peptide – 2 (GLP -2) treatment significantly decreases the intestinal permeability for bacterial translocation in acute pancreatitis.

Chronic Pancreatitis

In my study, I have come across 12 cases of chronic pancreatitis of which 6 cases were of obstructive type with cyst and 6 cases were non-obstructive type. The course of obstruction of pancreatic duct in my study was mainly calculi.

The other causes of obstruction are

1. Congenital (or) Acquired stricture of the pancreatic duct.
2. Pancreas divisum
3. Duct obstruction from tumours

4. Inflammation of the papilla of vater.
5. Protein malnutrition
6. Cystic fibrosis.
7. Hypercalcaemic states

Chronic pancreatitis can occur as a genetic condition transmitted as a Mendelian Dominant Trait. The condition is rare.

20 to 30% of chronic pancreatitis has no apparent cause so termed as idiopathic pancreatitis.

In my study, 75% of the cases of chronic pancreatitis were caused by alcoholism. Alcoholic pancreatitis generally occurs in patients who consume alcohol for at least 2 years and usually between 6 to 10 years.

Alcohol causes pancreatitis by any one of the following mechanism[9,12,13,14,15,16,29]

1. By inducing spasm of the sphincter of oddi thereby creating an obstruction to the outflow of pancreatic juice.

2. Alcohol is also a cellular metabolic poison and it has deleterious effects on the synthesis and secretion of digestive enzymes by the

pancreatic acinar cells. This causes an increase in the concentration of enzyme protein in pancreatic juice, and the eventual precipitation of this protein in the pancreatic ducts. Calcium may also precipitate with in the matrix protein plugs and obstruct the pancreatic ducts.

3. Alcohol increases the permeability of pancreatic ducts, there by initiate enzyme extravasations and cause pancreatic injury.

4. Alcohol significantly depresses pancreatic blood flow for several hours after ingestion. This may cause ischaemic injury to the gland.

In my study, most of the chronic pancreatitis patients had principal symptom of abdominal pain radiating to back or to left. Most of the patients were emaciated. Most of them were chronic alcoholic some were with diabetes mellitus. Repeated pain attack is characteristic of chronic pancreatitis. Pain free intervals become shorter and the pain eventually occurs everyday. Pain is mainly due to increased intraductal pressure up to 30 to 50cm of H_2O. (Normal up to 20cm of H_2O). Eating may increase the pain; so many patients avoid food and lose weight. Significant exocrine insufficiency will occur if 90% of secretary capacity of pancreas is lost. The major consequences are steatorrhoea and creatorrhoea. They may complain of bulk, offensive, fatty and oily stools.

Early in the disease serum amylase and lipase concentrations are elevated. As the disease becomes advanced, they often remain normal. Mild elevations of serum bilirubin, alkaline phosphatase and SGOT and mild depression of serum albumin can occur. Pancreatic function test are rarely indicated in chronic pancreatitis patients. ERCP and CT scan are more useful diagnostic tools.

In 30 to 50% of patients with chronic pancreatitis, plain X-ray abdomen reveals pancreatic calcifications. ERCP study provides important information about "ductal anatomy" that may influence a decision for surgery. e.g. For a dilated duct patient pancreatico jejunostomy is indicated. For a normal duct pancreatic resection is advisable. Strictures, cysts and ductal calculi may be seen. The characteristic "chain of lakes" seen. CT SCAN also useful for ductal anatomy study. Biliary dilatation and the level of bile duct obstruction is defined clearly. It also provides the most precise information about the size and configuration of the pancreas.

More than 2/3rd of chronic pancreatitis patients have diabetes mellitus. But the DM is usually mild and rarely associated with

ketoacidosis and vascular complications.

Non-operative management of chronic pancreatitis are

1. Control of abdominal pain
2. Treatment of endocrine insufficiency
3. Treatment of exocrine insufficiency

Control of abdominal pain

- Advise to stop alcohol intake (about 50%- of patients has some pain relief when they stopped alcohol)
- Advise to consume semi solid or liquid diets instead of solids.
- More carbohydrate.
- Less Fat & protein.
- H_2 receptor antagonists
- Oral pancreatic enzyme supplements
- Parentral somatostatin analogue to inhibit pancreatic secretion e.g. octreotide.
- Attempts to control pain often require early use of non-narcotic analgesics followed later by narcotic analgesics.

Treatment of exocrine insufficiency

- Dietary restriction of fat is important.
- Pancreatic enzyme replacement e.g. cotazym, ilozyme, viokase.

- H2 receptor blocking agent.

Treatment of endocrine insufficiency

- Mild elevations of blood sugar do not require treatment fasting level>250 mgm/dl should be managed with insulin.
- Maintain the fasting level around 200 mgm/dl

Surgical Treatment

In this study, out of 12 patients with chronic calculus pancreatitis, one patient presented with Pseudocyst with calculi in the pancreatic duct. The operative procedure was pancreatico lithotomy followed by Cystojejunostomy and jejuno- jejunostomy. Another five patients were treated surgically. The different procedures were as follows.

1.Pancreatico lithotomy followed by pancreatico jejunostomy and jejunojejunostomy - Two cases.

2.Pancreatico lithotomy followed by Roux-en- Y pancreatico jejunostomy and jejunojejunostomy - One case.

3.Pancreatico lithotomy, pancreatico jejunostomy, choledochojejunostomy, gastrojejunostomy - One case.

4.Pancreatico lithotomy, pancreatico jejunostomy, cholecysto jejunostomy, jejunojejunostomy - One case.

Operative management of Pseudocyst associated with chronic pancreatitis is effective with low morbidity and mortality rates[30]

The Primary goal of operative management is relief of pain; the secondary consideration is to preserve maximal endocrine and exocrine function. Prior to surgical intervention ERCP & CT study is must to study the ductal anatomy.

1. Drainage procedure - for dilated duct.
2. Pancreatic resection - for Normal (or) Narrow duct.

Drainage procedure (Pancreatico jejunostomy)

The main pancreatic duct has a normal diameter of 4 to 5mm in the head, 3 to 4 mm in the body, and 2 to 3mm in the tail. If the diameter is more than 7 to 8mm in body and head, a pancreatico jejunostomy **(Puestow procedure)** is technically feasible

- Operative mortality is about 4%
- Drain will close spontaneously.
- Patient may gain weight.
- Pain relieved in about 80 to 85% patients.
- Sometimes stenosis of pancreatico jejunal anastomosis may occur, then pancreatic resection is advisable.

Pancreatic resections

1.Pancreatico duodenectomy

2.Pylorus preserving pancreatico duodenectomy.

3.Pancreatic Head resection.

4. 95% pancreatectomy

5.Distal pancreatectomy

The main indication of pancreatic resection for a chronic pancreatitis patient is

1.To relief pain

2.Failure of drainage procedure

3.When the pathological changes involve one part of the gland and the rest is less diseased.

4.When the diagnosis between chronic pancreatitis and pancreatic cancer is in doubt

Pylorus preserving pancreatico duodenectomy

- The entire stomach, Pylorus and first 3 to 4cm of duodenum are preserved.
- Technically easier and more quicker.
- Preserve gastric function.

Pancreatic Head

Resection Indications

Most of the pathological changes involving.

- The head of pancreas
- 1/2 of duodenal compression.

  2/3rd of CBD compression.
- 1/4th of Portal vein compression.

Resected Structure

- Head of the pancreas is resected.
- Entire stomach and duodenum preserved.
- Body & tail of pancreas as well as a thin rim of pancreatic tissue in the 'C' loop of duodenum also remain.

Theoretical advantages

- Preservation of gastro duodenal and biliary continuity and function.
- Prevent the development of Diabetes

Ninety five percent distal pancreatectomy

Indications

1. If the entire pancreas is uniformly and severely diseased
2. Other operations have failed to provide relief.

Resected Structures.

1. Entire pancreas except for a thin rim of tissue that lies with in the 'C' loop of duodenum is removed.
2. Spleen is removed.

Distal Pancreatectomy

Definition: Resection of the pancreas reaching the left side of the superior mesenteric vein.

Indication:

1. Recurrent episodes of pancreatitis with multiple Pseudocysts in the tail of the pancreas and spleenic vein thrombosis.
2. Recurrent episodes of pancreatitis with a stricture in the main duct in the body of the pancreas and a dilated duct.

Resected Structures

- Variable amounts of tail (or) body of the pancreas are resected.
- Spleen is removed in most instances.
- Head of the gland is preserved.

Complications

1. Pancreatico jejunal fistula

2. Choledocho jejunal fistula

3. Injury to CBD

Pain is relieved in about 85% after resection. About 60 to 70% after drainage procedure.

The principle causes of deaths are

- Upper respiratory malignancies
- Malnutrition
- Complications of diabetes
- Suicide.

Pseudocyst of pancreas

In my study, out of 15 cases of Pseudocyst pancreas, most of the patients were chronic alcoholic. They presented with abdominal pain, vomiting and upper abdomen mass. Two patients developed Pseudocyst following a blunt injury abdomen and seven patients developed following an attack of acute pancreatitis. Two patients presented with enlarged GB with signs 'of obstructive jaundice.

Etiological factors of Pseudocyst are shown as follows

Etiological factors of Pseudocyst

1. Alcoholism - 10
2. Gall stones - Nil

3. Trauma - 2

4. Idiopathic - 2

5. Others - 1

All the patients have raised serum amylase level. Ranging from 426 U/L to 910 U/L Few patients show elevated alkaline phosphatase and serum bilirubin and elevated SGOT and SGPT levels. CT scan was useful in assessing the age of the cyst. ERCP can define the pancreatic ductal anatomy and cyst - duct communication. In some patients OGD was also done. OGD shows extra luminal compression of the stomach due to a lesion situated posterior to the stomach.

In my study the course of pancreatic Pseudocyst was analyzed. About 30% of fluid collections disappear spontaneously. Smaller cysts of size 4 to 5cm resolved spontaneously with conservative line of management. Only few patients with large size cyst treated surgically. I n this study in one patient Cystogastrostomy and in another patient Cystojejunostomy was done. The entire pseudopancreatic cyst is present in the region of Head of pancreas. Most were single and in only one patient multiple small cysts occurred.

Surgical treatment depends on the size, duration of the cyst, maturity of the cyst wall and presence (or) absence of infection of cyst

contents.

Large adherent retro gastric cysts are drained into the stomach, cysts with in the duodenal sweep are drained into the duodenal and in the tail of pancreas are drained into jejunum.

Complication of the internal drainage include

1.Gastro Intestinal Hemorrhage.

2. Cyst recurrence.

3 Sepsis.

Complications are more frequent following Cystogastrostomy than Cystojejunostomy. Cystogastrostomy is associated with shorter operative time and hospital stay, but a higher incidence of cyst recurrence.

The optimal therapeutic approach for multiple Pseudocysts is internal drainage. The options are internal cystostomy combined with enteric drainage for contiguous cyst and drainage by multiple Roux-en-Y Cystojejunostomy for non-contiguous cysts.

In this study external drainage of the pseudo pancreatic cyst was done in a patient, since it had ruptured into the peritoneal cavity. External

drainage is the operation of choice for

1.Infected cysts.

2.Those associated with hemorrhage

3.Free rupture into the peritoneal cavity.

4.Immature cysts that will not hold sutures.

It will be associated with a persistent pancreatic fistula, recurrence and mortality.

There are many non-surgical techniques for treating the pseudo pancreatic cysts.

They are

1.Needle aspiration

2.Percutaneous catheter drainage.

3.Percutaneous Cystogastrostomy.

4.Endoscopic cyst - enteric drainage.

5.Endoscopic transpapillary approach.

Percutaneous catheter drainage

Percutaneous catheter drainage is an excellent, initial option In

patients:

1.Who are critically ill

2.Who are high surgical risks

3.Who have infected Pseudocyst

Success rate is about 93%. Morbidity is about 13%

The drawbacks include

1.Occurrence of controlled external pancreatic fistula.

2.Risk of drain site infection.

3.High incidence of cyst persistence (or) recurrence.

Cysto gastric Drainage

Placement of a double pigtail stent using fluoroscopy with ultra sound guidance technique is an easy and safe alternative. Surgical drainage stents can be placed within a few weeks of development of the cyst and have been kept for a period ranging from 6 weeks - 10 months without any problems.

Endoscopic drainage is feasible and safe for selected cases of pancreatic Pseudocyst. The success of this procedure relies on close proximity of the cyst to bowel wall and the choice of drainage site. It can

be made only by Endoscopic demonstration of an obvious intra luminal bulge. This technique should not be used if the wall thickness is greater than 10mm

The serious complications are

1.Arterial bleeding.

2.Infections

3.Duodenal perforation.

Rupture of the Pseudocyst into the neighboring bowel is increasingly reported. Perforation into the colon carries a high morbidity and mortality where as fistula into the stomach (or) small bowel result in uneventful recovery.

Hemorrhage is an uncommon but serious complication of pancreatic Pseudocyst and the source of bleeding may be rupture of a pseudo aneurysm inside the Pseudocyst. Vascular involvement can be diagnosed by Ultra sonogram and angiography. Angiography embolizations, suture ligation of the bleeding vessel are the two lines of Management.

Several factors need to be considered in the choice of a suitable surgical method.

1.Site

2.The number of cyst.

3.Pancreatic endocrine and exocrine capacity.

4.Mechanical obstruction of the biliary tract gastric outlet.

5.Status of the main pancreatic duct.

Resection indicated when the patient has severe pancreatic endocrine insufficiency and when multiple Pseudocyst are located in the head of pancreas which are not suitable for internal drainage.

Ca Head of Pancreas

In this study I have come across 10 cases of Ca head of pancreas. Most of the patients had complaints of weight loss (90%), anorexia (90%), Jaundice (77%) and pain abdomen (60%). One patient with abdominal distension. Two patients were chronic alcoholic and chronic smoker. One patient with Diabetes.

Pancreatic ductal adenocarcinoma is about 90% of all malignant neoplasms of the gland. Highly fatal disease, 5-year survival rate is 1-2% only. Accounts for 10% all the cancer of digestive tract.

- Fourth most common cancer of all sites as a cause of death (after lung, colorectal and breast).
- More common in older people (Sixth to Eighth decade)
- Commoner in men than in women.

Pain and Weight loss are the two main consistent symptoms. It may be episodic and related to meals or it may become constant and chronic. Weight loss is severe and rapid. Haematemesis and melena are late features. Migratory Thrombophlebitis (Trousseau's sign) can be present.

I have not come across Ca body and tail of pancreas.

On examination epigastric mass is present. Sometimes liver & GB may be palpable (30%). Distant metastasis in the supraclavicular fossa (Troisier"s sign) may present Ascites may be present.

Possible Etiological factors of Ca Pancreas

- Cigarette smoking
- Alcohol abuse
- High Protein and high fat diet.
- Exposure to industrial carcinogens (eg) betanaphthylamine, benzidine.
- Chemists, workers in metal industries, coke and gas plant employees.
- Pancreatic cancer may develop in patients with a history of previous gastrectomy for peptic ulcer disease. Increased

production of N - nitroso compounds by bacteria that proliferate in the hypo acidic stomach could be responsible for both the development of gastric and pancreatic cancers.

- Mutant K ras gene on codon 12, Mutation of p53 gene on chromosome17.
- Diabetes Mellitus (Six fold risk in women not in men).
- Chronic pancreatitis[32]
- Hereditary pancreatitis, Von Hippel – Lindau's syndrome, Legneles syndrome – II.

Three patients came to hospital at a very late stage of cancer. The causes may be

1. The tumour is asymptomatic in early stage.
2. Patient delay.
3. Physician delay.
4. The patient may not have ready and easy access to competent diagnostic centers.

All the patients presented with liver function test abnormalities, including increased levels of total bilirubin, alkaline phosphatase and transaminases. Because of obstructive nature of these tumours, alkaline phosphatase is generally more elevated than the transaminases.

Pancreatic adenocarcinoma in humans, the specimens have over expression of Alpha – 1 anti trypsin, Glutothione- s- transferase and vascular endothelial growth factor[33].

Pancreatic cancer cells also express CCK – B/gastrin receptor[34] and gastrin precursor forms in most patients. Newer therapeutic strategies need to develop for the management of pancreatic cancer; targeting gastrin and its receptor may provide a novel treatment option.

Tumour associated antigen, CA 19-9 and CA 494 has a sensitivity and specificity for pancreatic cancers. CA 19-9 has been reported as 90% sensitivity and specificity. Unfortunately CA 19-9 is frequently normal in the early stages and cannot be used as routine screening test[35].

Ultrasonography and CT scan are more useful tests. Both test confirm the obstructive nature of jaundice by demonstrating dilated intra hepatic and extra hepatic bile ducts.

CT is more useful than USG in determining the level of obstruction, demonstrating the presence of pancreatic mass (as small as 1 cm in size) and detecting liver metastasis or local vascular invasion. MRI has no apparent advantage over CT.

ERCP (Endoscopic retro grade cholangio pancreatography) may be important if the differential diagnosis includes chronic pancreatitis.

Endoscopic examination is useful for Visualization of ampullary and duodenal carcinoma and for biopsy taking.

The percutaneous approach is usually technically easier with a dilated biliary tree and offers the' advantages of defining the proximal biliary system that will be used in reconstruction.

Duodenal drainage studies are recommended to obtain materials for cytological examination only. When ERCP has failed for technical reasons biliary drainage is useful in selected patients with advanced malnutrition. Sepsis and/or correctable medical conditions.

In my study all the patients with Ca pancreas are kept in a good state of nutrition and hydration with supplemental IV fluids, elemental diet and multivitamin as deemed necessary. Blood clotting deficiencies are corrected by giving Vit K for 3 days daily.

Even though the bilirubin levels of these patients are elevated to a maximum level of 15.8 mgm% we didn't performed biliary decompression procedures as cholecystostomy (or) T-Tube drainage of CBD. But ideally speaking for any patient with raised serum bilirubin

level more than 12mgm%, biliary decompression procedures either through Transhepatic route (or) Endoscopic approach should be done.

In this study we selected the patients for radical treatment only when they were found fit, i.e., not associated with distant metastasis, ascites, very old age.

In this study we did **Whipple's operation** (pancreatico duodenectomy). For four patients in Whipple's surgery we removed, head and neck of pancreas together with duodenum, distal half of stomach, lower CBD, Gall bladder and upper jejunum and as much of regional lymph nodes as possible.

Some articles described about the pylorus preserving technique in the Whipple's procedure[36] the classical Whipple's' reaction and pylorus preserving technique are equally effective with comparable and acceptable perioperative risks. Pylorus preserving resection offers the advantage of shorter operating times, less blood loss and decreased need for blood replacement and an increased ability to work after 6 months of surgery. Long-term results showed no differences in terms of overall survival tumor recurrence or quality of life; the procedures are equally effective for the treatment of pancreatic and peri – ampullary

malignancies. Reconstruction following pancreatico duodenectomy, pancreatico jejunostomy pancreatico gastrostomy. Some studies showed[37,38] pancreatico gastrostomy has fervor surgical complications.

The other surgical options are

1 Total pancreatectomy.

2. Regional pancreatectomy.

In total pancreatectomy, along with contents of Whipple's operation, the spleen, body and tail and regional lymph nodes are removed.

In Regional pancreatectomy along with the contents of total pancreatectomy, the transpancreatic portion of the portal vein, celiac axis, superior mesenteric artery and middle colic vessels are removed.

Among the three surgical options, total pancreatectomy with regional lymphadenectomy is recommended for the following reasons:

1. Pancreatic cancers are potentially multifocal in origin.
2. Gross and histological tumour spread has been documented at the line of resection.
3. Malignant viable cells are often present in the obstructed pancreatic ductal system and if the gland is divided, this may be a source of seeding for local recurrence.
4. The existence of lymphatic exchange between the head and the

body of the pancreas has been amply demonstrated.

5. Excision of the whole pancreas eliminates the risk of postoperative pancreatitis.
6. Preservation of endocrine (or) exocrine tissue is not sufficient justification for leaving part of the pancreas in situ. Over 80% of all pancreatic cancer patients are diabetic at the time of presentation.

Various modalities of treatment have been described regarding early cancers in periampullary regions. In an article published in 2005 in England[39]. Justifying the aggressive approach for pancreatic cancers. They came to a conclusion that, resection rate of 19% with increased median survival rate for pts with cancer by 8 months, more than that where not resected. The have studied 140pts and followed up for 6 months. Among them, 23 pts underwent whipple's procedure. 14 pts underwent triple bypass and 113pts underwent palliative treatment.

Their results were

Median survival – Triple bypass $\frac{5\ Months}{(Range\ 0.1\ to\ 20\ months)}$

Range 0.1 to 20 months 1

- Conservative treatment $\frac{3months}{(Range\ 0.1\ to\ 30\ months)}$
- Palliative chemotherapy $\frac{5months}{(Range\ 1\ to\ 30\ months)}$
- Whipple's procedure $\frac{13\ months}{(Range\ 5\ to\ 66\ months)}$

31 months for pts with clear resection

And negative nodes.

A cancer of the pancreas is considered unresectable if there are

1. Distant metastasis (liver or peritoneal).
2. Invasion of major vessels (portal vein, hepatic artery, superior mesentric vessels and celiac artery).
3. Any extension beyond the area of usual total pancreatectomy specimen. Puckering of the transverse mesocolon per se does not always indicate unresectability. It can be removed along with total pancreatectomy specimens.

In my study post operatively, two patients have developed renal failure symptoms and hypoproteinemia. Hypoproteinemia had been corrected by administration of injection Astymin and Human albumin and fresh blood. Patients with renal failure symptoms are treated with diuretics as per the advise given by the nephrologist.

The other expected complications are hemorrhage, Sepsis, Mesentric thrombosis, liver insufficiency, Myocardial infarction, cerebro vascular accident, congestive heart failure and pulmonary embolism. The incidence of hemorrhage is reduced by meticulous pre operative preparation and adequate replacement of blood and clotting factors during operation.

The re operation is indicated if:

1. If there is a reason to suspect a major bleeding site.
2. When clot accumulation in the abdomen causes distension and tamponade.
3. When a consumption coagulopathy is recognized.

One patient developed postoperative biliary leak. Patient was treated conservatively. Leakage from the biliary – enteric anastomosis or from the gastrojejunostomy is largely preventable by careful and proper consturction of anastomosis. Complications that are usually non fatal include pneumonitis, gastric retention, Paralytic ileus, bowel obstruction, wound injection, wound dehiscence, atrial fibrillation, faecal fistula and gastrojejunal fistula.

Post operatively the jaundice had reduced. Persistence of jaundice may be due to small bowel obstruction. The obstruction may be due to

recurrent tumour or simply due to adhesions. Laparotomy may be indicated to establish the diagnosis and to relieve the obstruction.

Leakage from residual pancreatic thump / pancreatico – enteric anastomosis are the major complication and death after pancreatic surgery due to corrosive nature of the pancreatic secretion. Synthetic somatostatin (14 Amino acid peptide hormone)[40] - octreotide, Lanreotide and vapreotide are used to decrease the pancreatic exocrine secretion. And use of somatostatin and its analogues appears to reduce the overall rate of post operative complications after pancreatic surgery.

Monitoring of recurrence:

To assess the recurrence of tumour some tumour markers are more useful. They are:

1. Pancreatic oncofetal antigen. (POA)
2. Carbohydrate antigen CA 19-9
3. Carcino embryonic antigen (CEA)

Serial monitoring of either marker may be useful in confirming the completeness' of surgical excision and in the detection of recurrent pancreatic cancer.

Adequate pancreatin tablets (Viollase, pancrease) must be taken

with each meal. The patient is advised to take a low fat diet in the from of frequent regular small meals.

The mortality rate in my study was 25%. One death was due to renal failure, and occurred after one month. Death is usually due to metastatic pancreatic cancer.

Place of the Whipple's operation.

If the surgeon cannot be sure of the exact site of origin of the tumour at operation, he may do standard Whipple's operation and immediate careful examination of the specimen by the pathologist and surgeon. If the tumour is not originating from the pancreas, the operation is adequate. If the surgeon and pathologist are not sure about the origin of the tumour, convert the procedure into total pancreatectomy.

In my study we have done palliative surgical procedures for three patients. The palliative procedure are done to relieve Jaundice, Pruritis, Impending cholangitis and for relief of duodenal obstruction.

Anterior. Gastrojejunostomy with jejunojejunostomy was done to relieve duodenal obstruction.

Cholecysto jejunostomy or hepaticojejunostomy to relieve jaundice.

Other Palliative measures to relieve the pain - - Celiac ganglion block with 50ml of 50% alcohol (or) with 20ml of 6% phenol.

Cordotomy, extensive sympathectomy and stereo tactic thalamotomy have all been tried. If a cancer in the head of pancreas a pancreatico gastrostomy with dilatation of the duct of Wirsung over a T-tube may be helpful.

In this study we have taken biopsy from unresectable tumour of pancreas in 3 patients and histopathological examination was done. After improving the general condition of the patient we gave chemotherapy.

Recent study showed Interferon based adjuvant chemoradition therapy improves survival after pancreatico duodenectomy for pancreatic adenocarcinom[41] apart from 5 FU & cisplatin.

When the patient is unfit or refuses operation an alternative method of palliating the obstructive jaundice is by Endoscopic sphincterotomy and placement of a biliary stent.

Conclusion

CONCLUSION

The incidence of pancreatic disease in this hospital is not very low. The Male : Female ratio is 2.5 :1. The most common pancreatic

disease in our hospital is acute pancreatitis with pseudo cyst following acute pancreatitis. Both the disease collectively contributing 48.5% of the total cases under study group. Chronic calculus pancreatitis and Ca head of pancreas contributing 27% each. Chronic alcoholism is the most common etiological agent for both acute and chronic pancreatitis. Chronic pancreatitis with ductal dilatation and severe symptomatic chronic pancreatitis without ductal dilatation can be better managed surgically. Pseudocyst of pancreas which was treated with internal drainage procedure had better results and early pseudocyst can be treated conservatively. In Carcinoma head of pancreas whipple's procedure carries 25% mortality in our hospital. In younger ages comparatively survival rate during and after surgery is better than older people.

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Annexure

MASTER CHART

Sl. No.	Name	Age	Sex	IP No.	Diagnosis	Treatment	Complication	Remarks
1.	Perumal	17	M	4754	Acute Pancreatitis	Conservative Line	Nil	NA
2.	Selvaraj	37	M	6212	Acute Pancreatitis	Conservative Line	Died	NA
3.	Roseline	20	F	11229	Acute Pancreatitis	Conservative Line	Nil	NA
4.	Saraswathy	43	F	15482	Acute Pancreatitis	Laparatomy & peritoneal Lavage	Wound gapping	Secondary Suturing
5.	Kannan	27	M	22106	Acute Pancreatitis	Laparatomy & peritoneal Lavage	Nil	NA
6.	Dhandapani	29	M	41671	Acute Pancreatitis	Laparatomy & peritoneal Lavage	Died on 4th POD	NA
7.	Chellamuthu	26	M	53109	Acute Pancreatitis	Laparatomy & peritoneal Lavage	Nil	NA
8.	Muthupandi	26	M	45844	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
9.	Balamurugan	29	M	53395	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
10.	Krishnasamy	37	M	2232	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
11.	Palaniappan	32	M	33089	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
12.	Gnana Prakash	19	M	66886	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
13.	Murugesan	35	M	57996	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
14.	Baskar	25	M	67512	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA

Sl. No.	Name	Age	Sex	IP No.	Diagnosis	Treatment	Complication	Remarks
15.	Balakrishnan	43	M	53338	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
16.	Arumugam	41	M	53138	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
17.	Muthusamy	31	M	58284	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
18.	Vinu	18	F	54418	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
19.	Veerannan	32	M	56393	Pseudocyst of Pancreatitis	Conservative Line	Nil	NA
20.	Rajamani	45	M	65495	Pseudocyst of Pancreatitis	External Drainage	Persistence of Leak up to 36 days	Treated conservatively
21.	Kumanan	35	M	61838	Pseudocyst of Pancreatitis	Cysto Gastrostomy	Nil	NA
22.	Rathinasamy	37	M	26338	Pseudocyst of Pancreatitis	Cysto Jejunostomy	Nil	NA
23.	Shanthamani	56	F	61459	Chronic Calculas Pancreatitis	Conservative Line	Nil	
24.	Vellingiri	35	M	44029	Chronic Calculus Pancreatitis	Pancreatico – Lithotomy with Cysto Jejunostomy with Jejuno Jejunostomy	Wound gapping	Secondary Suturing
25.	Saravanamuthu	39	M	25048	Chronic Calculus Pancreatitis	Pancreatico – Lithotomy with Pancreatico Jejunostomy with Jejuno Jejunostomy	Nil	NA

Sl. No.	Name	Age	Sex	IP No.	Diagnosis	Treatment	Complication	Remarks
26.	Subramani	31	M	45564	Chronic Calculus Pancreatitis	Pancreatico – Lithotomy with Pancreatico Jejunostomy with Jejuno Jejunostomy	Nil	NA
27.	Angammal	54	F	41682	Chronic Calculus Pancreatitis	Pancreatico – Lithotomy with Roux – N – Y - Pancreatico Jejunostomy with Jejuno Jejunostomy	Septicemia and Died on 6th POD	NA
28.	Vinoth Kumar	36	M	19306	Chronic Calculus Pancreatitis	Pancreatico – Lithotomy with Pancreatico Jejunostomy with Gastro jejunostomy with choledocho duodenos-tomy	Nil	NA
29.	Kumarasamy	39	M	41866	Chronic Calculus Pancreatitis	Pancreatico – Lithotomy with Pancreatico Jejunostomy with cholecysto jejunostomy with Jejuno Jejunostomy	Nil	NA
30.	Subban	49	M	61882	Chronic Calculas Pancreatitis	Conservative Line	Nil	NA
31.	Marudhachalam	47	M	55988	Chronic Calculas Pancreatitis	Conservative Line	Nil	NA
32.	Amminiammal	39	F	4815	Chronic Calculas Pancreatitis	Conservative Line	DM was developed	NA
33.	Thangamani	37	F	57342	Chronic Calculas Pancreatitis	Conservative Line	Nil	NA

Sl. No.	Name	Age	Sex	IP No.	Diagnosis	Treatment	Complication	Remarks
34.	Ramanammal	61	F	589562	Chronic Calculas Pancreatitis	Conservative Line	Nil	NA
35.	Aruchamy	57	M	0844	Ca Head of Pancreas	Whipple's Procedure	Bile Leak	Treated Conservatively
36.	Kaliappan	53	M	6031	Ca Head of Pancreas	Whipple's Procedure	ARDS	Treated with Ventilatory Support
37.	Kuppusamy	55	M	16728	Ca Head of Pancreas	Whipple's Procedure	Septicemia	Died of Renal Failure at the end of 1st Month.
38.	Vasanthi	42	F	61112	Ca Head of Pancreas	Whipple's Procedure	Nil	NA
39.	Rajammal	45	F	12542	Ca Head of Pancreas	Triple Bypass	Nil	NA
40.	Pappathy	51	F	36811	Ca Head of Pancreas	Triple Bypass	Nil	NA
41.	Subramani	29	M	22214	Ca Head of Pancreas	Triple Bypass	Nil	NA
42.	Rajalakshmi	69	F	64114	Ca Head of Pancreas	Open biopsy and chemo therapy	Died on 4th month	NA
43.	Sivakami	62	F	73412	Ca Head of Pancreas	Open biopsy and chemo therapy	Not came for follow-up after 4th month	NA
44.	Subbanna Goundar	68	M	7676	Ca Head of Pancreas	Open biopsy and chemo therapy	Not came for follow-up	NA
45.	Sulaiman	69	M	9191	Ca Head of Pancreas	CT guided FNAC with chemo therapy	Not came for follow-up	NA

Coimbatore Medical College Hospital , Coimbatore -18

Department of Surgery

VARIOUS PANCREATIC LESIONS

(An analytical study of 45 cases)

Academic period May 2004 – January 2006

PROFORMA

Name:	Age:	Sex:	IP No.:
Occupation:		Income:	
Series No. :	Unit:		
D.O.A.:	D.O.S.:	D.O.D:	

History of Present Illness:

1. H/o Abdomen pain -
2. H/o Vomiting -
3. H/o Fever -
4. H/o Trauma -
5. H/o Hiccup -
6. H/o Anorexia -
7. H/o Jaundice -
8. H/o Weight loss -
9. H/o Abdominal distention -
10. Others -

Past History:

History of similar episode in the past -

Other medical illness -

H/o Diabetes Mellitus -

Personal History:

Diet	-	Yes / No
H/o Smoking	-	Yes / No
H/o Alcohol	-	Regular
		Occasional

Family History

Similar illness in their family members -

General Physical Examination

Conscious	-	Yes / No
Oriented	-	Yes / No
Dehydrated	-	Yes / No
Pallor	-	Yes / No
Icterus	-	Yes / No
Pedal Oedema	-	
Temperature	-	
Pulse	-	

BP -

Respiratory rate -

S_{PO2} % -

Examination of Abdomen

Epigastric mass -

Hepatomegaly -

Gall bladder -

Ascities -

Per Rectal Examination

CVS -

RS -

Pre Operative Diagnosis

Investigations

Urine	Albumin
	Glucose
	Bile salts
	Bile pigments
	Urobilinogen
	Deposit

Complete Hemogram	
	HB %
	TC
	DC
	ESR
	Bleeding Time
	Clotting Time
	Platelet count
	Hematocrit
Blood Glucose	
Blood Urea	
Serum Creatinine	
Serum Electrolytes	Na^+
	K^+
	HCO_3^-
	Cl^-
Liver Function test	
	SGOT
	SGPT

	SAP	
	GGT	
	Serum Bilirubin	Total
		Direct
		Indirect
	Serum protein	Total
		Albumin
		Globulin
	Serum amylase	
	Serum lipase	
	Serum calcium	
Blood Grouping and typing		
Plain Chest X-Ray		
Plan X-Ray Abdomen	Erect	
	Supine	
Ultra Sound Abdomen	First	
	Second	
	Third	
CT Abdomen		

Treatment

Pre Operative Management

Operation

Per Operative Findings and Surgery

Post Operative Period

Follow up

HPE Reports